IN THE SPECIFICATION

Kindly enter the following paragraphs.

Page 1, insert as a separate paragraph following the title:

This is a continuation of Appln. No. 09/770,405, filed January 29, 2001, pending; which is a continuation of Appln. No. 09/413,807, filed October 7, 1999, now U.S. Patent 6,251,406; which is a continuation-in-part of Appln. No. 09/288,861, filed April 9, 1999, now U.S. Patent 6,458,368; which is a national-stage of Appln. No. PCT/GB97/02740, filed October 7, 1997; the entire contents of which are hereby incorporated by reference in this application.

Page 10, eighth paragraph:

Plasmid pFS14nsd HPV16-L1 was constructed by exchanging in the plasmid pFS14 NSD (54) the hepatitis B nucleocapsid gene (HBcAg, NcoI-HindIII fragment) for a NcoI-HindIII fragment encoding the HPV16-L1 open reading frame. The HPV16-L1 NcoI-HindIII fragment was generated by Polymerase Chain Reaction (PCR) using the baculovirus expression plasmid pSynwtVI HPV16 114/B-L1+L2 (23) as a template with a 28 mer containing a NcoI site 5'-GGG*CC**ATG**G*CTCTTTGGCTGCCTTAGTGA-3' (SEQ ID NO:1) and a 27 mer containing a HindIII site 5'-GGG*AAGCTT*CAATACTTAA-GCTTACG-3' (SEQ ID NO:2). The final construct containing the Tac promoter places the HPV16-L1 **ATG** at position +8 relative to the Shine-Dalgamo sequence and introduces a change in the second amino acid which becomes an alanine instead of the serine encoded by the original sequence. Sequencing of the entire L1 open reading frame was carried out (Mycrosynth AG) and no further nucleotide change was observed. Plasmid pFS14nsd HPV16-L1 was amplified in *E. coli* JM105 and then electroporated as described previously (50) into bacterial strain CS022. This strain is derived from the ATCC 14028 strain, into which the pho-24 mutation was introduced by P22 transduction, resulting in attenuation in both virulence and survival within macro-phages in vitro ($PhoP^c$ (35)). The resultant recombinant strain is called $PhoP^c$/HPV hereafter.